REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. AGENCY USE ONLY *(Leave blank)*	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	2005	Book Ch.-Exercise Testing & Exercise Pres, etc.

4. TITLE AND SUBTITLE
Environmental Factors for Exercise Testing and Exercise Prescription

5. FUNDING NUMBERS

6. AUTHOR(S)
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8. PERFORMING ORGANIZATION REPORT NUMBER
MISC02-15

9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)
Same as #7 above

10. SPONSORING / MONITORING AGENCY REPORT NUMBER

11. SUPPLEMENTARY NOTES

12a. DISTRIBUTION / AVAILABILITY STATEMENT
Distribution is unlimited.

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*
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14. SUBJECT TERMS
Temperature regulation, energy balance, thermoregulation

15. NUMBER OF PAGES
24

16. PRICE CODE

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
Unclassified	Unclassified	Unclassified	Unclassified

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Environmental Factors for Exercise Testing and Exercise Prescription

Samuel N. Cheuvront, Michael N. Sawka, and Kent B. Pandolf

The clinical use of exercise testing to evaluate an individual's cardiorespiratory reserve and to enable the appropriate prescription of aerobic activity has attained wide medical acceptance. The physiologic stress from the metabolic intensity imposed by exercise, however, is only one consideration in testing and prescription. An equally important stress is that imposed by the environment (heat, cold, altitude, and air quality). Both exercise and environment alter the physiologic responses of the cardiorespiratory system. These environmental factors, either singly or in combination with exercise, can result in potentially hazardous health conditions.

Temperature Regulation and Energy Balance

The control mechanisms of temperature regulation for dealing with overcooling (cold stress) are not as effective as those for regulation against overheating (heat stress). Consequently, humans are thought of as tropical animals. Such behavioral modifications as increased food intake and adequate clothing are typical human reactions to overcooling. In contrast, control mechanisms for thermoregulation are primarily structured to protect the body against overheating. This fact becomes readily apparent when one considers that greater variations in core temperature (T_c) than $\pm 4°C$ are associated with reduced physiologic and psychologic performance, whereas deviations of about $+6°C$ or $-12°C$ from $37°C$ (normal T_c) are usually lethal. Combined exercise and heat stress can result in greater strain on the thermoregulatory system than either stress alone, whereas exercise stress may counteract cold stress and result in less overcooling. Fortunately, the human thermoregulatory system has a remarkable ability to maintain physiologic control through appropriate adjustments over an extremely wide range of different heat productions, heat losses, and environmental temperatures.

Physical exercise dramatically alters the rate of metabolic energy (heat) production (M), with resultant physiologic adjustments for heat loss. During exercise, T_c initially increases rapidly and then increases at a reduced rate until heat loss equals heat production and essentially steady-state values are achieved (1). The T_c increase represents storage of metabolic heat produced as a byproduct of muscle contraction. At the onset of exercise, metabolic rate increases immediately, while thermoregulatory responses for heat loss respond more slowly. The classic energy balance equation for evaluating heat gain or heat loss from the body is $S = M - (\pm W) + Q_s \pm K \pm (R + C) - E$, in which S = rate of body heat storage; M = rate of metabolic energy (heat) production estimated from measured oxygen uptake ($\dot{V}O_2$); W = mechanical work, either

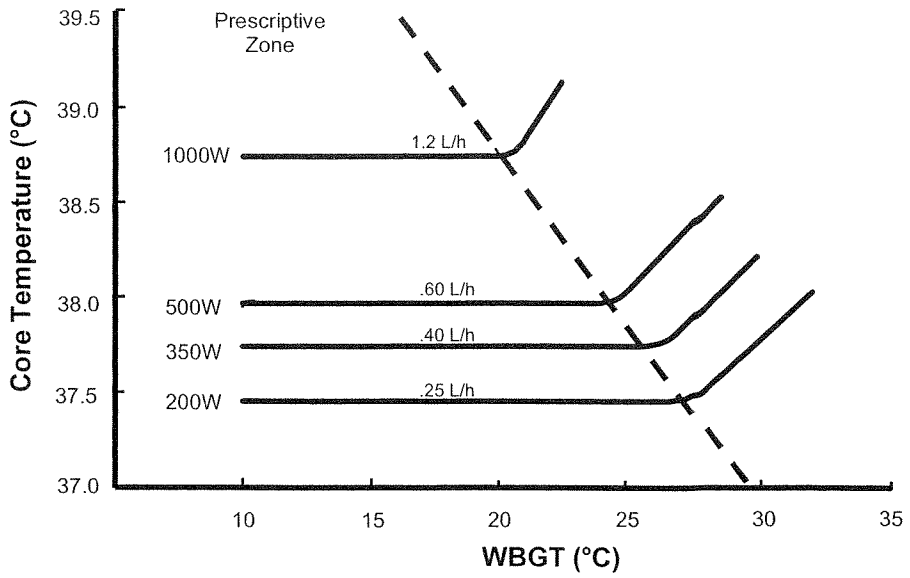

FIGURE 7-1 Avenues of heat exchange during exercise for a wide range of ambient temperatures. (Modified from Sawka MN, Wenger CB, Pandolf KB. Thermoregulatory responses to acute exercise-heat stress and heat acclimation. In: Fregly MJ, Blatteis CM, eds. Handbook of Physiology, section 4, environmental physiology. New York, Oxford University Press, 1996:157–185.)

concentric (positive) or eccentric (negative) exercise; Q_s = rate of solar radiative energy absorbed (differentiates between heat loss in sunlight and that lost from skin in an environment without solar flux); K = rate of conduction (important only when in direct contact with an object, e.g., clothing, or a substance, e.g., water); R + C = rate of radiant and convective energy exchanges; and E = rate of evaporative loss. The three major physical avenues of heat loss are radiation, convection, and evaporation. The thermoregulatory effector responses that enable dry (radiative and convective) and evaporative heat loss increase in proportion to the rate of heat production. Eventually, these mechanisms increase heat loss sufficiently to balance metabolic heat production, allowing a steady-state T_c to be achieved, so long as the environment allows.

Steady-state T_c increases in proportion to metabolic rate during exercise (2–5). Although this relationship between metabolic rate and T_c is adequate for a given person, it does not always hold for comparisons among different people. The use of relative intensity ($\%\dot{V}O_{2max}$), rather than absolute metabolic rate (absolute intensity), removes most of the intersubject variability for the T_c elevation during exercise (6,7). Therefore, relative exercise intensity is an important factor when prescribing exercise. Figure 7-1 illustrates that the magnitude of T_c increase during steady-state exercise at low (200 W) to very high (1000 W) metabolic rates is often independent of a wide range of environments (with low humidity) (1,2). This is true, however, only within a climatic "prescriptive zone" (2) that narrows as metabolic rate increases.

Ultimately, the combination of the level of exercise and the particular environmental conditions determines the rate of sweat production and dictates the required rate of evaporative cooling (E_{req}). The maximal evaporative capacity of the environment (E_{max}), however, determines the maximal possible evaporative loss. Figure 7-2 illustrates heat exchange data during exercise (~650 W) in a broad range of climatic conditions (5 to 36°C dry bulb temperatures with low relative humidity, % rh) (1,8). The difference between metabolic rate and total heat loss represents the energy used for mechanical work and heat storage. The relative contributions of dry and evaporative heat exchange to total heat loss, however, vary with climatic conditions.

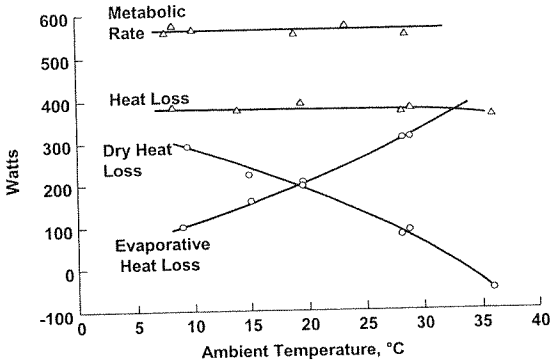

FIGURE 7-2 T_c response and required sweating rates for exercise in relation to metabolic rate and environment (Modified from Sawka MN, Wenger CB, Pandolf KB. Thermoregulatory responses to acute exercise-heat stress and heat acclimation. In: Fregly MJ, Blatteis CM, eds. Handbook of Physiology, section 4, environmental physiology. New York, Oxford University Press, 1996:157–185.)

As ambient temperature increases, the gradient for dry heat exchange is less and evaporative heat exchange is more important. When ambient temperature equals mean skin temperature ($\overline{T}_{sk}$), evaporative heat exchange accounts for virtually all heat loss. For individuals exercising outdoors, the solar radiant load becomes an important consideration.

It is beyond the scope of this chapter to present a detailed essay on temperature regulation and exercise. The reader is directed to other reviews and books (9–13).

Exposure to High Environmental Temperatures

HUMAN PHYSIOLOGIC RESPONSES TO HEAT

The two physiologic responses primarily concerned with dynamic regulation against overheating are skin blood flow and sweating. The main function of the former is to transport heat from the deep body to the surface. Sweat glands produce and secrete sweat required for evaporative cooling at the skin surface. During exercise, regulation by these systems is increasingly challenged when air temperature (T_a) rises from temperate (~22°C) to hotter conditions.

Metabolic (exercise-induced) and environmental heat stress can result in normal or expected physiologic responses to the particular stress, but may also produce a variety of abnormal heat disorders. Although the primary purpose of this section is not to detail the pathologic manifestations of excessive heat exposure, a few comments on how to prevent these heat disorders are warranted. Exercise-induced heat exhaustion can be minimized by providing proper heat acclimation, by grading the exercise to consider hot climate extremes, and by avoiding sudden postural changes or maintenance of upright static exercise. Heat cramps and salt-depletion heat exhaustion can occur when fluid and electrolyte losses are profound. These disorders are most prevalent in chronically hot climates, particularly when food and water intakes are limited or restricted. Avoid drinking in excess of sweat losses to avoid hyponatremia (14). Fluid needs rarely exceed 8 to 12 $L \cdot day^{-1}$, even in very hot climates (15,16). Salt tablets should not be used and, if necessary, supplemental sodium can be easily obtained by salting foods (16). Anhidrotic heat exhaustion, which is related to reduced functional sweating, has been linked to such skin disorders as heat rash and sunburn; both disorders have been associated with exercise-heat intolerance, as depicted by elevations in T_c and reductions in performance time (1,17). Heat intolerance was seen with as little as 20% of the body surface involved and persisted up to 3 weeks after the clinical rash had resolved. Pandolf et al. (18) also showed that both sweating sensitivity and sweating rate were reduced by mild sunburn during exercise in a hot/dry environment (49°C, 20% rh). Thus, both of these common hot-weather ailments can significantly impair thermoregulation. Heat rash is prevented by drying the skin when possible and by wearing clean, dry clothing that allows unimpeded evaporation. Sunburn is reduced by wearing proper clothing, by exercising during hours of minimal solar load (before 10 AM or after 4 PM), or by using an appropriate sunscreen. Heat hyperpyrexia and exertional heat injury/heat stroke present dramatic elevations of T_c, usually in the range of 41 to 42°C. The former disorder is usually characterized by lower T_cs in this range, with the individual still capable of sweating. The latter condition presents higher T_cs and generalized anhidrosis. Elevations in T_c, however, may not be causally related to these disorders, because both

competitive distance runners and patients with passively induced hyperthermia tolerate T_cs of 41 to 42°C with minimal side effects (19,20). Both heat hyperpyrexia and heat stroke can be prevented by adapting exercise to the climate, by ensuring proper heat acclimation, and most importantly by screening for a past history of heat illness.

THERMOREGULATORY AND CARDIOVASCULAR ADJUSTMENTS TO EXERCISE IN DRY AND HUMID HEAT

Effects of Solar Heat Load

The importance of quantifying the physiologic effects of solar radiation becomes apparent when considering exercise prescriptions for outdoor environments. The effect of simulated solar heat load in hot/dry (40°C, 32% rh) and hot/wet (35°C, 75% rh) environments has been reported for heat-acclimated men while walking at 1.34 $m \cdot sec^{-1}$ (0 and 5% grade) (21). Evaluation of solar load by copper manikin predicted the delivery of an effective 300 W (seminude) and 120 W (clothed) of radiant heat load to the skin. Individuals were evaluated with or without this solar load while wearing shorts, socks, and running shoes (seminude) or a slightly heavier clothing ensemble. After 100 min of exercise, physiologic responses were greater while exercising with solar load (range of mean differences; heart rate (HR), 22–42 $beats \cdot min^{-1}$; rectal temperature (ΔT_{re}), 0.45–1.48°C; sweat rate ($\dot{m}_{sw}$), 145–314 $g \cdot m^{-2} \cdot h^{-1}$. Nielsen et al. (22) measured net solar heat gain (short- and long-wave radiation gains − long wave radiation losses) during 2 hours of light (92 W) cycle ergometer exercise performed outdoors between July and September during maximal solar zenith angle hours (10 AM to 4 PM). With temperate climates (21–25°C), direct solar heat amounted to ~22% of the total heat load (22). Taken together, these studies suggest the need for altering exercise prescriptions during outdoor activity, especially on hot, clear days.

Role of Cardiorespiratory Training and Fitness

The importance of training and cardiorespiratory fitness on physiologic responses to exercise in the heat and on the rate of heat acclimation is controversial, but there are several detailed reviews (23–25). Although most authors agree that training in a cool environment improves exercise-heat tolerance, the degree of improvement is controversial. To achieve optimal gains, researchers suggest using intensive interval or continuous training at an intensity >50% $\dot{V}O_{2max}$ (26–29). Improvement in heat tolerance produced by mild-to-moderate training at <50% $\dot{V}O_{2max}$ is questionable (30). It seems that training must exceed 1 week, but the best improvement reportedly occurs after 8 to 12 weeks (26,27). It seems that training should increase $\dot{V}O_{2max}$ by 15 to 20% to improve tolerance. Improvement in exercise-heat tolerance after appropriate training appears to apply to both dry and wet heat. Persons with high $\dot{V}O_{2max}$ values and such athletes as marathoners (whose endurance training causes high levels of body hyperthermia and regulatory sweating) seem to be at an advantage.

Another debatable issue is whether $\dot{V}O_{2max}$ is related to improved exercise-heat tolerance or to a faster rate of heat acclimation. Two authors using different climates independently report that an individual's $\dot{V}O_{2max}$ accounts for 42 to 46% of the variability that determines T_c level during 3 hours of exercise in the heat of the acclimation day for a plateau in T_c (31,32). Other authors report insignificant relationships (33–35). Most studies in which a lack of relationship was shown, however, evaluated relatively few subjects or homogeneously fit subjects. The $\dot{V}O_{2max}$, per se, may not be important, but the physiologic adaptations associated with various fitness levels may play a vital role in determining exercise-heat tolerance. Therefore, improved aerobic fitness by endurance training is associated with significant elevations in T_c during training to improve exercise-heat tolerance (23). Athletes training in cold water have lower heat tolerance than other athletes of similar fitness (27) or experience no thermoregulatory improvement during exercise-heat stress despite significant (15%) training improvement in $\dot{V}O_{2max}$ (36). Thus, training that improves fitness without substantial elevations in T_c and sweating may not improve heat tolerance.

Advantages of Heat Acclimation

Repeated exercise-heat exposure results in a gradual acclimation with improved exercise-heat tolerance (37). The physiologic improvements seen during the first 4 days are dramatic, and acclimation is

virtually complete after about 10 days. During acclimation, the major physiologic changes are an earlier onset and increased rate of sweating, lowered HR_e, and lowered internal body temperature during exercise in the heat (38). These changes result from many potential mechanisms, including improved sweating, more total body water and plasma volume, higher venous tone from cutaneous and noncutaneous beds, increased activity of the adrenopituitary system, and lowered metabolic demands from repeated exposures (38). No single cause explains the adaptive process, as acclimation probably results from the interplay of many mechanisms.

The full development of exercise-heat acclimation need not involve daily 24-hour exposure. A continuous, daily 100-min exposure can produce an optimal response (1,38). The acclimation response is somewhat specific to the particular climatic condition and exercise intensity. It appears to be well retained for 2 weeks after the last heat exposure but is rapidly lost during the next 2 weeks (1,38). Some authors report greater retention of acclimation benefits in physically trained persons than in sedentary individuals (31,38).

Clothing Interaction

During exercise in the heat, black clothing and possibly other dark colors are usually associated with a greater solar radiative absorption than white or lighter colors. Generally, clothing serves as a physical barrier that reduces heat exchange by radiation and convection and simultaneously lowers the maximal evaporative exchange to the environment. The more impermeable the clothing (e.g., a sweat suit or rubberized suit), the greater the reduction in evaporative heat loss and the associated rise in cardiovascular and thermoregulatory strain. For comfort, cotton clothing is generally more effective than polyester during exercise in the heat. In hot/wet environments, when T_a is less than $\overline{T}_{sk}$, exercising individuals should wear the least amount of clothing possible. When T_a exceeds $\overline{T}_{sk}$, additional clothing may protect from the ambient heat load but will interfere more with body heat loss. It is advisable to wear loose-fitting clothing that allows greater airflow between skin and the environment, with resultant greater evaporative cooling. In hot/wet or hot/dry environments, a thin layer of white clothing markedly reduces the solar heat load and should be worn when exercising under the sun. Associated concepts have been recently reviewed in detail elsewhere (39).

IMPACT OF HEAT ON EXERCISE TESTING AND EXERCISE PRESCRIPTION

Sports and occupational medicine communities commonly use wet bulb globe temperature (WBGT) as an empirical index to quantify climatic heat stress (40–42). It was originally developed for light-intensity exercise (45). Outdoor WBGT = 0.7 natural wet bulb + 0.2 black globe + 0.1 dry bulb; indoor WBGT = 0.7 natural wet bulb + 0.3 black globe. WBGT is used to decide the permitted physical activity level and strategies to minimize risk of heat injury. High WBGT values can be achieved by high humidity (43), as reflected in high wet bulb temperature, or through high air (dry bulb) temperature and solar load (44), as reflected in black globe temperature.

The American College of Sports Medicine (ACSM) position stand on preventing thermal injuries during distance running is based in part on the WBGT, which may also be adaptable for exercise testing and prescription (46). With a WBGT >28°C (82°F), the ACSM suggests that prolonged exercise be curtailed or rescheduled until a lower WBGT is prevalent. The ACSM proposes posting large signs to alert individuals of the existing risk of thermal stress using four categories. Very high risk is associated with a WBGT >28°C (82°F), high risk is 23 to 28°C (73 to 82°F), moderate risk is 18 to 20°C (65 to 73°F), and low risk is <18°C (65°F) (46). These WBGT values are representative for persons in running shorts, shoes, and a T-shirt. However, because WBGT does not consider clothing or exercise intensity (metabolic rate), it cannot predict heat exchange with the environment (40). Therefore, different clothing systems necessitate further adjustments in the WBGT values associated with each level of risk. Finally, the ACSM recommends that when environmental heat stress is prevalent, all exercise should begin in the early morning (before 8 AM) or in the evening (after 6 PM)

to lessen the effects of solar load and high temperatures.

Exercise Performance

The exercise prescription often uses a target HR that is within "safe" limits to provide a beneficial training stimulus. Heat stress increases HR independently as a result of increased skin blood flow and volume, reduced cardiac filling (lower end-diastolic volume), and temperature effects on pacemaker cells (38). During submaximal exercise in the heat, cardiac output ($\dot{Q}$) can be higher, the same, or lower than in temperate conditions. At very low exercise intensities (<20% aerobic power), $\dot{Q}$ is elevated to increase skin blood flow. With high-intensity (>70% aerobic power) or prolonged exercise, $\dot{Q}$ cannot typically be sustained, despite significant tachycardia, because blood is displaced to the skin, with a resultant drop in venous return (12). However, $\dot{Q}$ may increase under the same circumstances in highly fit athletes who can maintain stroke volume at an increased HR (47).

In young male subjects after 30 min of moderate exercise (40% $\dot{V}O_{2max}$), HR increases predictably ~1 beat·min^{-1} for each 1°C increase in T_a (dry heat) above temperate levels (24 vs. 44, 55°C) (48). Recently, more quantitative predictions of equilibrium exercise HR response were made for typical hot/dry (40°C, 20% rh) and hot/humid (35°C, 75% rh) environments in contrast to a temperate (21°C, 50% rh) environment (49–51). It was assumed that the subject weighed 70 kg, wore shorts and a T-shirt, exercised at a high metabolic rate (700 W), and was not heat acclimated. Compared with values in a temperate climate, HR was 30 beats·min^{-1} higher (~1.5 beats·min^{-1} per 1°C rise in T_a) in the hot/dry and 50 beats·min^{-1} higher (~3.5 beats·min^{-1} per 1°C rise in T_a) in the hot/wet climate. Thus, whereas adjustments are needed in dry climates, particular concern should be given to the target HR in humid climates. These prediction equations should be used to *individually* adjust the exercise prescription target HR when considering the particular environmental conditions, exercise intensity, and clothing interactions (49,50). A mathematical model based on the above prediction equations has been developed to prognosticate human performance in the heat and may be useful to establish exercise prescriptions (52). This prediction model calculates sustainable exercise–rest cycles, maximal single exercise time (if appropriate), and associated water requirements. Target HR responses should be adjusted and reevaluated periodically to consider seasonal effects.

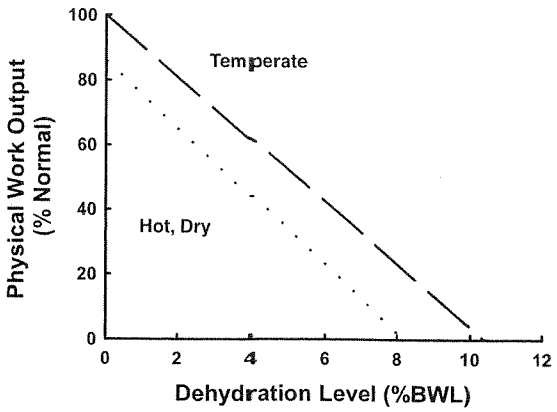

FIGURE 7-3 Effects of air temperature and dehydration on submaximal work performance. (Modified from Sawka MN, Young AJ. Physical exercise in hot and cold climates. In: Garrett WE, Kirkendall DT, eds. Exercise and Sport Science. Philadelphia: Lippincott Williams & Wilkins, 2000:385–400.)

Figure 7-3 provides a literature compilation regarding the effects of climatic heat stress (moderate-to-high risk categories) and dehydration on aerobic exercise capabilities (13). This analysis is based on highly motivated and heat-acclimated persons. Note that heat stress reduces physical exercise (submaximal) capabilities by ~20% of that under temperate environmental conditions. The combination of heat stress and moderate (4% body weight) dehydration can reduce work capabilities by ~50% of what is expected for fully hydrated individuals in temperate conditions (13). Mechanisms that explain this observation include increased cardiovascular strain and psychologic factors that independently or in combination diminish the will to exercise (13).When physical activity is expected in the heat, especially on a clear day (solar load) or under humid conditions, reduced exercise intensity and target HR are essential. The impact of dehydration alone on exercise testing and prescription is discussed below, along with recommendations for fluid replacement.

Special Populations

Heat tolerance is generally reduced in older individuals, and cardiovascular strain is greater because of reduced fitness and the inability to partition and regulate blood flow effectively (53). While hot environments predictably increase cardiovascular strain, as measured by changes in HR, these changes are not necessarily associated with pathologic outcomes in persons with compromised cardiovascular health. Physiologic responses to 30 min of static–dynamic work (shoveling) were compared in 10 men with stable ischemic heart disease in warm (29°C), temperate (24°C) and cold (−8°C) environments (54). While HR was higher during warm weather, no subject reported angina, and no abnormal ST-segment changes were observed during any trial. Similarly, Sheldahl et al. (55) reported higher HR without myocardial ischemia or reduced ejection fraction in coronary heart disease (CHD) patients (≥6 weeks post-cardiac event) after 60 min of cycling exercise in warm (30°C) conditions than in temperate (22°C) conditions. While these observations provide valuable insight for exercise prescription, when the level of environmental stress necessitates precaution during exercise for healthy populations, exercise for special populations should also be curtailed.

Exposure to Low Environmental Temperatures

HUMAN PHYSIOLOGIC RESPONSES TO COLD

Humans generally rely on behavioral strategies (e.g., clothing, shelter) for protection from cold. When behavioral thermoregulation provides inadequate protection, physiologic defenses to combat cold are elicited in the form of alterations in peripheral circulation that reduce heat loss and increase heat production (56). The initial response to cold stress is peripheral vasoconstriction to reduce heat loss from the deep body to the periphery. The next major response involves greater skeletal muscle activity, or shivering, which increases metabolic heat production (3–4 times the resting level) (13). These physiologic responses may influence or be influenced by the physiologic responses to exercise (13,57), which easily results in a 10-fold increase in heat production and can effectively counteract moderate cold stress.

A variety of factors can alter cold tolerance (56). Body size and shape alter heat loss to the environment. For a given cold stress, smaller persons (children and women) need a relatively greater heat production to maintain thermal equilibrium because of their larger surface area-to-mass ratios (58). Similarly, shorter persons who weigh the same as taller persons lose less heat, principally because they have less exposed surface area (shorter arms, legs, and trunk). Thickness of subcutaneous fat deposits is also important. Generally, subcutaneous fat is an effective insulator, with greater amounts being negatively related to the fall in T_{sk} or T_c (56). The advantages of increased subcutaneous fat are apparent for all types of cold exposure, but particularly cold water immersion (58). Skeletal muscle mass is another important factor because it can generate heat via voluntary (exercise) or involuntary (shivering) contraction. Respiratory heat and water loss can be substantial during exercise in the cold. Most of the loss occurs as evaporation to humidify the very dry cold air that is inhaled. Upper-airway temperatures may fall substantially during exercise if extremely cold air is breathed, but the lower respiratory tract and deep body temperatures are unaffected (59).

Most body heat loss in cold environments occurs via conductive (K) and convective (C) mechanisms. When ambient temperature is colder than body temperature, the resulting thermal gradient favors body heat loss. While the pathophysiology of cold injury is not a major consideration of this chapter, a few comments on preventing cold disorders during exercise are warranted. Cold disorders can be categorized by nonfreezing (muscle cramps, chilblains, and immersion/trench hand or foot) or freezing (frostnip and frostbite) cold injuries and whole body hypothermia. A major consideration in prevention is an adequate definition of the cold stress (i.e., the particular ambient temperature and wind velocity), as well as the presence of sweat on the skin or in the clothing. For maximal protection, clothing should be layered and thick and *must* be kept dry. To prevent cold injuries, heat supply to the periphery must be enhanced. Auxiliary heating of the extremities through battery-charged gloves and socks should help maintain safe temperatures. Alcoholic drinks produce peripheral vasodilation and therefore promote heat loss, enhancing the risk of

hypothermia (60). Gradual acclimation to cold over 2 to 3 weeks may induce peripheral changes that help the individual to resist local cold injury (56). Because exercisers seldom spend much time outdoors and because exercise increases heat production, the risk of whole body hypothermia is minimal, provided the individual does not stay in the cold in a sweat-soaked or rain-soaked state.

CARDIOVASCULAR AND THERMOREGULATORY RESPONSES TO EXERCISE IN THE COLD

Cooling the body can result in marked systemic alterations (56). The most apparent alteration is perhaps peripheral vasoconstriction, which leads to a reduction in local circulation and reduced perfusion of various vascular beds, producing vascular stasis and local tissue anoxia. There is an initial paradoxical increase in HR, pulmonary ventilation, and mean arterial pressure. As deep body temperature drops, however, HR, ventilation, and blood pressure (BP) fall. Neurohumoral activation leads to release of the anterior pituitary hormones and catecholamines to conserve body heat. Other than physical exercise, however, the major reflex response for increased heat production involves higher muscle tone and shivering. In certain circumstances, periodic oscillations in $\overline{T}_{sk}$ occur reflecting transient changes in blood flow to superficial capillaries of the limbs (cold-induced vasodilation) (61). This nervous reflex appears to act as a primary mechanism to protect peripheral tissue from freezing injury or to maintain dexterity.

Mild-to-moderate cold stress of a less prolonged nature (~1 hour) is more likely to be encountered by the exerciser than is prolonged severe cold stress. Even less severe cold stress alters cardiovascular performance and produces circulatory changes that augment myocardial oxygen requirements, thus placing some individuals at added risk. For instance, mild cold stress (15°C) during rest and light exercise causes a consistently higher total peripheral resistance (TPR), higher systemic arterial pressure, and greater left ventricular work in individuals with and without CHD (62). This higher TPR occurs in the absence of reflex bradycardia and $\dot{Q}$ is not altered by the cold. Exposure of the face to a moderate cold stress (4°C) has been associated with bradycardia, resulting from a vagal reflex through trigeminal nerve stimulation (63). Sympathetic nervous system stimulation produces a rise in systolic (SBP) and diastolic BP (DBP). Thus, facial exposure to cold winds and whole body exposure might precipitate angina in an individual with CHD. Exercise would only accentuate this risk by demanding further increases in left ventricular work and myocardial oxygen demand.

COLD ACCLIMATION AND ADAPTIVE HABITUATION

Compared with chronic heat stress, physiologic adjustments to chronic cold exposure are less pronounced, slower to develop, and less practical in terms of relieving thermal strain and preventing cold injury. Cold acclimatization or acclimation must be differentiated from adaptation or habituation. Acclimatization and acclimation are functional alterations established over days or weeks in response, respectively, to either complex, natural environmental factors or artificially controlled, usually simple environmental factors (64). In contrast, adaptation suggests physiologic changes that develop over generations and are genetically transmitted to help promote survival in hostile environments (64). Habituation to cold stress seems to be associated more with nervous system regulation. Physiologic adjustments during human adaptation or acclimation to cold have been reviewed elsewhere (56).

Human Acclimation and Adaptive Habituation to Cold

Human thermoregulatory adaptations to chronic cold exposure are more modest and less understood than adaptations to chronic heat (56). Where chronic heat exposure induces a fairly uniform pattern of adjustments, chronic cold exposure induces three different patterns of adaptation. Habituation is characterized by blunted physiologic responses during cold exposure. Metabolic adaptations are characterized by enhanced thermogenic responses, and insulative adaptations are characterized by enhanced body heat conservation (56).

Brief, intermittent cold exposures can induce habituation of shivering and vasoconstrictor responses to cold, even when very limited body surface areas

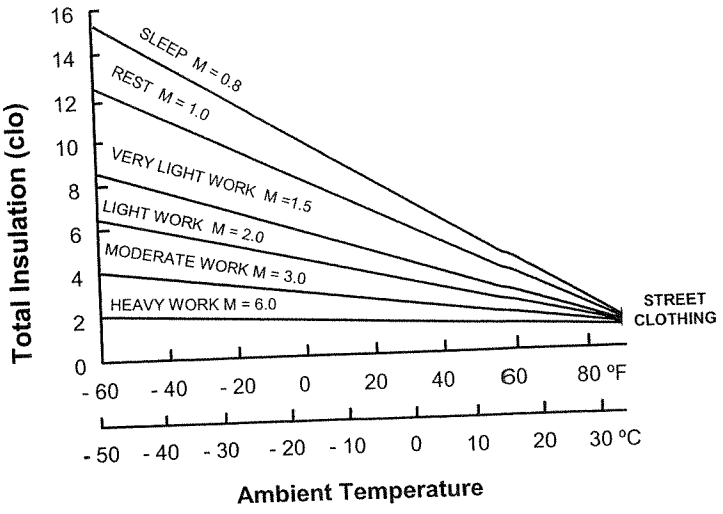

FIGURE 7-4 Total clothing insulation (clo) necessary for thermal comfort at various air temperatures and metabolic rates (M, METS). (Modified from Sawka MN, Young AJ. Physical exercise in hot and cold climates. In: Garrett WE, Kirkendall DT, eds. Exercise and Sport Science. Philadelphia: Lippincott Williams & Wilkins, 2000:385–400.)

are exposed and whole body heat losses are probably negligible (56). More pronounced physiologic adjustments are observed only when repeated cold exposure causes significant body heat loss, such as insulative adjustments in response to repeated cold exposure that are too severe to be offset by increased metabolic heat production (i.e., when cold causes a significant drop in T_c) (56). The possibility that an enhanced thermogenic capability can develop in humans in response to chronic cold cannot be dismissed. It is tempting to speculate that the stimulus for this metabolic pattern of cold adaptation is prolonged periods in which significant body heat loss is experienced, but under conditions in which body heat production increased sufficiently to prevent a significant decline in deep body temperature. This speculation is not unjustified, since the metabolic pattern of cold adjustments has only been reported in studies in which acclimatization or acclimation was induced by exposure to such conditions, i.e., prolonged exposure to moderately cold air (56).

Clothing Interaction

Using proper clothing should be stressed when prescribing exercise in cold air. Clothing should be thick and multilayered, while providing adequate ventilation to keep it dry. All areas of the body (particularly face, ears, neck, hands, and feet) should be covered adequately and kept warm. However, many outdoor winter sports and recreational activities require participants to disregard heavy insulative garments in favor of less restrictive clothing for freedom of motion. Clothing insulation needed for warmth and comfort in cold environments is much higher during rest and light activity than during strenuous activity (Fig. 7-4) (13). The solution is to dress in multiple clothing layers that allow insulation to be adjusted according to activity level, such that heat storage and sweating can be minimized. When exercise is stopped, any accumulation of sweat in clothing will compromise its insulative value and facilitate heat loss by conduction, convection, and evaporation. By the same token, rain or accidental immersion will drastically magnify these effects via disproportional moisture absorption and greater body surface area contact with wet clothing. Proper cold weather clothing should be wind resistant, but must provide adequate ventilation to reduce sweat accumulation. Proper protection of the face and extremities may be of greater concern.

IMPACT OF COLD ON EXERCISE TESTING AND EXERCISE PRESCRIPTION

Although exercise increases metabolic heat production, it also facilitates heat loss by convective heat transfer from the body core to the skin. When low ambient temperatures are combined with considerable wind speeds, convective heat losses become magnified, especially when exercise intensity is

reduced because of fatigue, and the risk of hypothermia can occur in environmental temperatures of 10 to 12°C WBGT (50–54°F) (9,46). In fact, it is more common to see exhaustive collapse in distance runners suffering from hypothermia than from hyperthermia (65). The ACSM recommends canceling outdoor distance running events when air temperature is below −20°C (−4°F) (46).

While no single cold stress index integrates all the effects of environment on heat loss, the wind chill index (WCI) is widely accepted and used (66). The WCI estimates the environmental cooling rate from the combined effects of the wind and air temperature and is useful for guiding decisions about conducting or canceling outdoor activities, although some limitations exist (see ref. 67 for review). Water has a much higher thermal capacity than air, and the cooling power of the ambient environment is greatly enhanced under cold/wet conditions. Thus, even with relatively mild water temperatures, swimmers and outdoorsmen who wade streams can lose considerable body heat. Special precautions should therefore be used for those at risk, especially those who use swimming as a form of exercise. Swimming in unheated or improperly heated pools and in the ocean (50% of which is <20°C) should be approached with extreme caution.

Exercise Performance and Special Populations

Although extremely cold temperatures can affect muscle function and reduce work performance (57), sustained exercise generally results in adequate heat production and maintenance of muscle temperature. Cutaneous cold receptor stimulation can, however, lead to increased TPR, arterial pressure, myocardial contractility, and cardiac work during rest or exercise (62,68,69). These altered responses were observed even during very mild cold stress (~15°C) (62). Even localized facial exposure to moderate cold (4°C) has been associated with vagally mediated reflex bradycardia and sympathetically increased SBP and DBP (63). Thus, localized and more total body cold strain could lower the threshold and provoke an attack of angina pectoris in individuals with CHD because of an increase in TPR and arterial pressure and the consequent augmentation of myocardial oxygen demands. There is no evidence that coronary vasoconstriction per se contributes to developing myocardial hypoxia during cold stress. During cold exposure, exercise could further increase the work of the heart through added myocardial oxygen requirements. Thus, an individual with CHD is at even greater risk because of far less functional myocardial reserve capacity. The importance of these observations for cold weather exercise prescription is only precautionary for compromised, but low-risk populations. While information is limited, one study of low-risk patients with stable ischemic heart disease reported higher SBP and DBP responses to 30 min of static–dynamic exercise in a cold environment (−8°C) than to the same activity in warmer (24–29°C) environments (54). This finding is consistent with greater TPR and potentially greater left ventricular work, but no adverse ST-segment changes (or any other ECG symptomatology) were observed.

A screening test for individual cold sensitivity may become necessary if it is suspected that a high-risk individual will be exposed to even mild cold stress (~15°C or less). The cold pressor test, which was designed to detect persons who were potentially hypertensive, might be used effectively to classify individuals in terms of reactivity to a cold stimulus (70,71). During immersion of the hand in cold water, sympathetic activity can be graded by the rise in SBP and DBP, elevation of HR, and degree of systemic vasoconstriction, as implied by the reduction in $\bar{T}_{sk}$ of the immersed hand. Extreme reactivity would contraindicate exercising in the cold for those with signs of CHD. As an additional caveat, long-term breathing of cold air can also increase respiratory passage secretions and decrease mucociliary clearance (72), potentially producing airway congestion, impairing pulmonary mechanics, and increasing the difficulty of any given exercise bout.

Exposure to Terrestrial Altitude

SHORT- AND LONG-TERM PHYSIOLOGIC ADAPTATIONS TO ALTITUDE EXPOSURE

Acute and Subacute Exposure to High Altitude

The most prominent adaptation to acute altitude exposure is an increased pulmonary ventilation ($\dot{V}_E$) at any given exercise $\dot{V}O_2$ (73,74). Hyperpnea

results in an elevated respiratory exchange ratio (R), reflecting increased CO_2 elimination from the lungs, and an associated rise in blood pH (73). In contrast to long-term altitude exposure, increased $\dot{Q}$ is noted during the first few days at altitude and is attributed mainly to a higher HR (74–76). These cardiopulmonary alterations are, in part, an attempt to enhance oxygen transport and delivery and thus help compensate for the lower oxygen pressure. These compensatory mechanisms are not adequate, however, and exercise performance is usually severely limited relative to the other two adaptive stages. Other factors related to performance alterations are uncompensated alkalosis, altered endocrine function, body fluid changes, and disturbed metabolic function (73). During short-term exposure, mountain sickness can develop with such symptoms as headache, lethargy, drowsiness, fatigue, sleep disturbances, loss of appetite, digestive disorders, and, less frequently, nausea and vomiting (75,77). A more serious but rare disorder is high altitude pulmonary edema, associated with fluid accumulation in the lungs (75,77).

The functional changes observed during subacute altitude exposure are also transient (74). The subacute stage is associated with adaptations that increase the oxygen-carrying capacity of the blood. Arterial oxygen content is restored (increased hematocrit) to near sea level values secondary to a decreased plasma volume (73–76,78,79). Although this hemoconcentration may increase blood oxygen content, these alterations are also associated with small decreases in convective blood flow secondary to increased blood viscosity (73,75,76). The second major change seen involves decreased submaximal and maximal $\dot{Q}$, attributable to a reduction in SV (74), probably as a result of decreased plasma volume and compromised ventricular filling (80). A further transient increase in $\dot{V}_E$ (particularly at heavier exercise intensities) is seen during this adaptive stage and is referred to as *ventilatory acclimatization* (73,76,79). At a constant $\dot{V}O_2$, $\dot{V}_E$ at altitude may increase by nearly 100%.

Long-Term Exposure to High Altitude

Although the functional alterations seen during long-term exposure (2–3 weeks) are similar to those seen during subacute adaptation, Hannon and Vogel state that these functional alterations differ in four major respects (74). Long-term adaptations to altitude develop more slowly, are not transient, are probably associated with all system components, and produce functional system capabilities that exceed those seen with subacute adaptation (74).

Although initiated during the acute adaptive stage, the effects of increased erythropoiesis become most pronounced during the chronic stage (78). While increasing hemoglobin content, the greater number of circulating red blood cells further decreases blood flow and increases blood viscosity. Thus, cardiac work may be increased at any given $\dot{Q}$ (73). Biochemical and histologic changes become more apparent after long-term exposure, which may facilitate the increase of either oxygen conductance or transport or both. For example, compared with the other two adaptive stages, there is a more pronounced capillary density, an increased myoglobin content in the skeletal muscles, and other modifications of enzymatic activity that may facilitate oxygen transport after long-term exposure to hypoxia (81). These functional alterations require different time periods for complete long-term adaptation and may necessitate significantly longer time periods when exercise responses are considered (74).

IMPACT OF ALTITUDE ON EXERCISE TESTING AND EXERCISE PRESCRIPTION

Maximal Exercise Performance

Many studies show a progressive reduction in $\dot{V}O_{2max}$ with increasing altitude. Fulco et al. (82) reported this relationship for altitudes from 580 to 8848 m; a slightly modified version is illustrated in Figure 7-5. The regression line in Figure 7-5 predicts a 5 to 10% decline in $\dot{V}O_{2max}$ for every 1000 m of ascent (76), although wide variability exists due to multiple factors, and disproportionately larger decrements may be observed above 6300 m (82). Smaller (1–7%) reductions in $\dot{V}O_{2max}$ are reported for well-conditioned athletes beginning at 350 to 580 m (82,83). The decrement in $\dot{V}O_{2max}$ is larger for highly fit men than for less fit men, presumably due to inherent pulmonary gas exchange limitations ($\dot{V}_A/\dot{Q}$ mismatch; $\dot{V}_A$-alveolar ventilation) exacerbated by hypoxia (82). This hypothesis is consistent with the $\dot{V}_A/\dot{Q}$ mismatch and reduced arterial oxygen saturation (%SaO_2) observed for athletes with

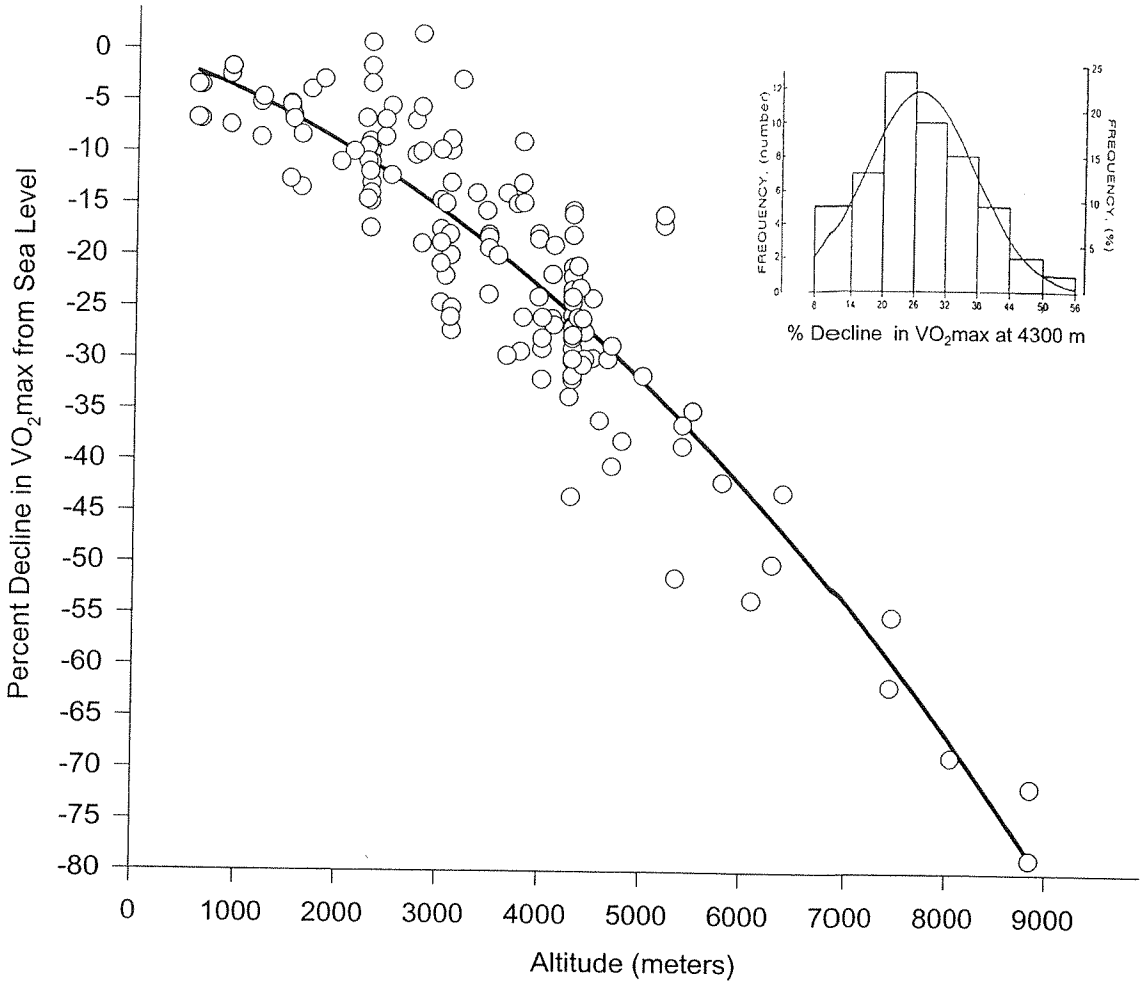

FIGURE 7-5 Effects of reduced partial pressure of oxygen at altitude on maximal oxygen uptake ($\dot{V}O_{2max}$) when expressed as a percentage of $\dot{V}O_{2max}$ at sea level. (Modified from Fulco CS, Rock PB, Cymerman A. Maximal and submaximal exercise performance at altitude. Aviat Space Environ Med 1998;69:793–801.) *Inset:* Distribution of decrement in percentage $\dot{V}O_{2max}$ at 4300 m (Modified from Young AJ, Cymerman A, Burse RL. The influence of cardiorespiratory fitness on the decrement in maximal aerobic power at high altitude. Eur J Appl Physiol 1985;54:12–15.)

elite $\dot{V}O_{2max}$ values (84,85). Although reportedly (86) sea level $\dot{V}O_{2max}$ is a poor predictor of $\dot{V}O_{2max}$ decline at altitude (4300 m), this is probably explained by the fact that only subjects with subelite $\dot{V}O_{2max}$ values were studied. These data illustrate that the decline in $\dot{V}O_{2max}$ at 4300 m is normally distributed for a range of fitness abilities usually encountered in an exercise testing arena (36–60 $mL \cdot kg^{-1} \cdot min^{-1}$) (86). Any decrease in $\dot{V}O_{2max}$ will persist at a given altitude, provided the level of cardiorespiratory physical fitness is not altered. Upon return from higher altitude, $\dot{V}O_{2max}$ returns to the sea level value so long as muscle mass is maintained (87).

The greatest reduction in $\dot{V}O_{2max}$ usually occurs during the first few days of altitude exposure, with a small but significant increase seen with persistent residence (76,81). The early reduction is generally

attributed to the lowered arterial oxygen content and the persistent decrease to a reduction in $\dot{Q}_{max}$ (81,88). Reduced $\dot{Q}_{max}$ results from a decrease in SV combined with a lower maximal HR (89). Maintenance of plasma volume at altitude may maintain ventricular filling pressure and prevent a decrease in SV (90,91). Increased vagal tone (90,92) and reduced cardiac β-receptor responsiveness (81,93) have been implicated in the lower maximal HR. The reductions in $\dot{V}O_{2max}$ and $\dot{Q}_{max}$ at 4300 m are not equal (94), suggesting the involvement of such factors as ventilatory, hemodynamic, and metabolic changes that together decrease $\dot{V}O_{2max}$. Reviews of the cardiovascular and peripheral adaptations to exercise at high altitude are found elsewhere (76, 81).

Submaximal Exercise Performance

For any given power output (PO) at sea level, $\dot{V}O_2$ is similar at altitude (95). But because submaximal exercise performance is closely tied to $\dot{V}O_{2max}$, any PO corresponds to a greater relative exercise intensity at altitude. Therefore, at the same absolute sea level PO, submaximal exercise performance is reduced at high altitude. For events lasting 20 to 30 min and 2 to 3 hours at sea level, Fulco et al. (82) report that absolute performances at 2000 m altitude would be impaired by 5% and 10 to 15%, respectively, i.e., performance decrements are proportional to altitude and exercise duration (82).Limitations are more profound for short- than long-term altitude exposure (96). Whereas the limitations imposed by the cardiovascular system primarily alter maximal exercise responses at altitude, differences in the use rate of glycogen stores and anaerobic metabolism influence submaximal exercise responses during short- and long-term altitude exposure (81). The physiologic alterations at altitude that help to improve tissue oxygen delivery when oxygen availability is reduced may be classified into three separate stages: acute, subacute, and chronic adaptation. Acute adaptation occurs during the first 72 hours of exposure; subacute adaptations are seen over the next 10 to 11 days; and chronic adaptations are observed after 2 weeks of exposure. Although the adaptations are presented as three distinct stages, they really represent a continuum of change with considerable individual overlap and variability.

Submaximal and maximal exercise performance is reduced at altitude (2000–5000 m) and an exercise prescription should be adjusted accordingly. Absolute exercise levels should be reduced 5 to 10% for each 1000 m ascent above sea level if exercise is to be performed at the same relative intensity (81). Using the information in Figure 7-5, many individuals can adjust exercise appropriately when the prescription is based on relative exercise intensity.

Special Populations

Grover implies that as long as persons with cardiovascular disease recognize their limitations and maintain exercise intensity within these limits, cardiac performance will not be compromised (75). According to Levine et al. (97), >5 million people over the age of 60 visit high altitudes annually. Because an aged population is at increased risk for cardiovascular disease, interest in the response of the elderly to hypobaric hypoxia has grown. The clinical responses of 97 older men and women (59–83 years) were studied over 5 days at moderate altitude (2500 m). Although CHD was present in 20% of subjects, no adverse signs or symptoms were observed during casual activity (98). Therefore, persons with asymptomatic cardiovascular disease can safely visit moderate altitudes. Levine et al. (97) studied the response to exercise in 20 veterans (mean age, 68 years) at sea level, 2500 m (acute), and 2500 m (chronic, 5 days). The double product (HR × SBP) that produced 1 mm of ST-segment depression was 5% lower at 2500 m (vs. sea level), but stabilized after 5 days at altitude (97). They concluded that (*a*) performance at altitude was predictable on the basis of sea level performance and (*b*) those with CHD should initially limit activity and allow several days to acclimatize to altitude (97). A study of the effects of moderate altitude on patients with CHD and impaired left ventricular function found that exercise at 2500 m was terminated more often because of dyspnea than at sea level but was otherwise tolerated well and without any adverse events (99).

In contrast, exposure to high altitude places greater demands on the right ventricle of the heart, resulting in increased total cardiac work (i.e., greater coronary blood flow), particularly during exercise (100). Some authors report such ECG disturbances during exercise as atrial and ventricular ectopic beats and prolonged QT interval, as well as diphasic, inverted, or flat T wave and lowered ST segment in nonadapted individuals at altitudes of 3000 to 5000 m (101). These results may have been confounded by the addition of cold stress.

Whether these responses are particularly important and potentially dangerous for persons with CHD may depend on the altitude studied, as well as on the seriousness and stability of the cardiovascular condition. At the very least, persons with advanced or unstable disease should (*a*) limit sojourns to low-moderate altitude near adequate facilities for cardiovascular care, (*b*) ascend to altitude slowly, and (*c*) limit activity to below symptom-limiting sea level intensities (102).

Exposure to Atmospheric Pollutants

Little is known about the effects of single agents or combinations of atmospheric air pollutants on either submaximal or maximal exercise performance. Even less is known about the particular concentration levels and critical exposure durations of air pollutants necessary for decrements in exercise performance. Of the air pollutants, only carbon monoxide (CO) has been evaluated with any thoroughness regarding cardiovascular responses to exercise. In healthy individuals, CO does not alter submaximal exercise responses, but slightly reduces maximal exercise performance. Individuals with cardiovascular impairments show marked decreases in submaximal exercise time to angina onset while breathing CO (Table 7-1).

Among the remaining air pollutants evaluated, the photochemical oxidants ozone (O_3), nitrogen dioxide (NO_2), and peroxyacetylnitrate (PAN) and the sulfur oxides, represented by sulfur dioxide

TABLE 7-1 EFFECTS OF AIR POLLUTANTS ON EXERCISE PERFORMANCE OF NORMAL AND CARDIOVASCULAR/PULMONARY-IMPAIRED INDIVIDUALS

Subject Group Air Pollutant •Exercise Intensity	Performance Decrement	No Effect	Selected References
Normal population			
CO			
• Submaximal		X	106, 107
• Maximal	X		109, 110
O_3			
• Submaximal		X	111, 112
• Maximal	?		114, 115
NO_2			
• Submaximal		X	103, 117
• Maximal	?		103
PAN			
• Submaximal		X	106, 116
• Maximal	?		103, 108
PANCO			
• Submaximal		X	106, 116
• Maximal	?		103, 108
SO_2			
• Submaximal	?		103
• Maximal	?		103
Cardiovascular/pulmonary-impaired population[a]			
CO	X		120, 122
O_3	X[b]		103, 123, 124, 125
NO_2	?		103
PAN	?		103, 108
PANCO	?		103, 108
SO_2	?		103

[a] Denotes submaximal exercise effects only.
[b] Applies to pulmonary-impaired (COPD and asthma) populations only.

(SO_2) and PANCO, have no demonstrable cardiovascular effects during submaximal exercise in healthy individuals. There are questionable effects, however, in healthy individuals during maximal exercise and in CHD patients during submaximal exercise. Table 7-1 summarizes the effects of air pollutants on exercise performance for healthy and cardiovascularly impaired individuals. The reader is directed for further information to reviews by Raven (103), Gong and Krishnareddy (104), and Carlisle and Sharp (105).

IMPACT OF ADVERSE AIR QUALITY ON EXERCISE TESTING AND EXERCISE PRESCRIPTION

Healthy Individuals

CO impairs cardiovascular function during exercise by binding with hemoglobin (COHb) to impede oxygen transport. Minimal impairment of cardiorespiratory function and no major performance decrements were observed in healthy individuals at COHb levels below 15% at submaximal exercise intensities of 35 to 60% $\dot{V}O_{2max}$ of short or prolonged duration. HR, however, increased significantly, and added respiratory distress was noted at ~70% $\dot{V}O_{2max}$ (106,107). In contrast, $\dot{V}O_{2max}$ was inversely related to CO concentration (107–110). The critical level at which COHb significantly influences $\dot{V}O_{2max}$ is reportedly 4.3%, but even lower levels (2.7%) have been associated with significant decrements in maximal exercise time (108–110).

The photochemical oxidants seem to cause lung and respiratory tract dysfunction, with questionable effects on the cardiovascular system during exercise (103). Of these, O_3 has been studied most thoroughly. During submaximal exercise (40–70% $\dot{V}O_{2max}$) after or during exposure to 0.37, 0.50, or 0.75 ppm O_3, no significant alterations in submaximal $\dot{V}O_2$, HR, or $\dot{V}_E$ were reported (111,112). Other measurements of pulmonary function, however, were somewhat disturbed (112). No significant differences in pulmonary function were reported between continuous and intermittent submaximal exercise (0.30 ppm O_3)when the total effective dose was the same (113). The limited observations concerning a true decrement in $\dot{V}O_{2max}$ with O_3 exposure are debatable. Some authors report no change in exercise capacity or $\dot{V}O_{2max}$ while breathing filtered air (FA) or 0.15 or 0.30 ppm O_3 and others show an 11% lower $\dot{V}O_{2max}$ while exposed to 0.75 ppm O_3 than while exposed to FA (114,115). Thus, the critical concentration level for reduced performance is questionable, particularly during maximal exercise. Raven suggests that exercise performance decrements can be predicted as a function of $\dot{V}_E$ and exposure time (103).

Of the other oxidants, PAN and PANCO have been evaluated during exercise stress (106,108, 116). During submaximal exercise (35% $\dot{V}O_{2max}$) lasting 3 hours while breathing 0.24 ppm PAN or PANCO (50 ppm; COHb, 4–6%), no remarkable changes in cardiorespiratory function were observed in younger (18–30 years) or older (40–55 years) subjects (106,116). Forced vital capacity was reduced 4 to 7% in the younger subjects with PAN, but the significance is questionable. No significant reductions in $\dot{V}O_{2max}$ were reported while breathing these same concentrations of PAN or PANCO (108,110). It seems premature to conclude that PAN or PANCO have no adverse effects on exercise performance, particularly at maximal levels. The concentration of PAN (0.24 ppm) in these few experiments may be at or slightly below the threshold level needed for demonstrable physiologic effects.

Even less is known about exercise-related effects of another oxidant (NO_2) and the sulfur oxides as represented by SO_2. Concentration levels of 0.62 ppm of NO_2 were evaluated after 2 hours of exposure at 40% $\dot{V}O_{2max}$ with no significant alterations in cardiorespiratory function (117). No significant changes were found in pulmonary function of male athletes at 50% $\dot{V}O_{2max}$ during 30 min of exercise (0.18 and 0.30 ppm of NO_2) (118). At low submaximal exercise intensity (only double the resting $\dot{V}_E$), exposure to 0.37 ppm of pure SO_2 did not change ventilatory function after 2 hours of intermittent exercise exposure (119). Maximal exercise responses to these two pollutants have not been reported. A synergistic effect between SO_2 and O_3 has been described in terms of a greater reduction in ventilatory function during exercise, raising the question of possible synergism among other pollutants (119). There is little information concerning the responses to long-term or prolonged exposure (>4 hours) to these pollutants at various concentrations during exercise.

Special Populations

Of the pollutants, only CO has been directly evaluated during exercise in cardiovascularly impaired individuals. It has been suggested that there is a relationship between CO and advanced development of CHD and that CO in the presence of significant CHD hastens myocardial infarction, angina pectoris, or sudden death. In a study of 10 CHD patients, exposure to heavy freeway traffic for 90 min increased COHb to an average of 5.08%, causing a decrease in exercise time to angina onset and significant reductions in SBP and HR at the onset of angina (120). Ischemic ST-segment depressions were noted in 3 of 10 patients while breathing freeway air, in contrast to no abnormalities during freeway driving while breathing compressed, purified air. In two studies, each involving 10 patients with documented angina, exercise angina onset time was determined while breathing 50 ppm of CO (COHb, 2.7%) for 2 hours or 50 ppm of CO (COHb, 2.9%) and 100 ppm of CO (COHb, 4.5%) for 4 hours (121,122). During both studies at either CO concentration, the average exercise times to onset of angina were lower than the values while breathing compressed, purified air. Duration of angina was significantly prolonged after breathing 100 ppm of CO, but not after breathing 50 ppm of CO (121). Generally, deeper and more prolonged ST-segment depressions were noted after breathing CO (121). Thus, CHD patients are at significant risk during exercise at low levels of COHb (2.5–3.0%). Raven concludes that "the cardiac-impaired exercising patient will be placed at increased risk of incurring additional coronary events if ambient levels of CO in the inspired air are capable of causing a rise of blood COHb levels above 1.5 to 2.0%" (103).

One might expect that individuals with cardiovascular or pulmonary disorders would be at risk during exercise while exposed to oxidants and sulfur oxides because of their limited cardiovascular or pulmonary reserve capacities. However, patients with documented CHD exposed to 0.20 or 0.30 ppm of O_3 during 40 min of treadmill exercise were no more susceptible to ozone toxicity than clinically normal persons (123). In contrast, persons performing light exercise with documented chronic obstructive pulmonary disease (COPD) exposed to 0.24 ppm of O_3 or documented asthma at 0.16 ppm of O_3 demonstrated significant lung dysfunction (124,125).

Submaximal exercise performance of persons without cardiovascular disorders does not appear to be compromised within the limits of the particular concentration levels evaluated for the different air pollutants. At near-maximal or maximal exercise, performance does appear limited for these same people when exposed to the same pollutants. In contrast, individuals with impaired cardiovascular systems are at even greater risk during submaximal exercise and exposure to CO; tissue hypoxia and myocardial ischemia may result from the binding of CO to hemoglobin. Evidence is limited concerning the adverse effects of the oxidants and sulfur oxides on the cardiovascular system of these compromised individuals during exercise. The oxidants (O_3, NO_2, and PAN) and sulfur oxides, which increase airway resistance because of reflex bronchoconstriction, place individuals with lung and respiratory tract disorders such as COPD and asthma at particular risk during exercise. Obviously, individuals with disorders of both the cardiovascular and respiratory systems are at even greater risk during exercise when exposed to these pollutants.

Most studies on exercise performance and air pollutants involve short-term exposure to particular pollutant(s). Little is known about long-term exposure to adverse air quality and the impact on exercise performance. For many air pollutants (e.g., CO), it takes 8 to 12 hours or longer to reach an equilibrium state between the inspired concentration and the level within the body (121). Thus, the cardiovascular or pulmonary burden should be even greater during exercise after prolonged exposure to adverse air quality. The possible synergism between various pollutants (in terms of added cardiorespiratory distress for acute or prolonged exposure) has not been evaluated during exercise and may further tax the cardiac or pulmonary reserves. Given the many unanswered questions concerning adverse air quality and those specifically at risk during exercise, supervising professionals who prescribe/lead exercise programs should act conservatively when this environmental stress is considered (103).

The current primary U.S. government concentration standards for these common atmospheric air pollutants are 9 ppm/8 h and 35 ppm/1 h of CO exposure; 0.12 ppm/1 h of O_3 exposure; 0.05 ppm/

1 year of NO_2 exposure; 0.14 ppm/24 h of SO_2 exposure, and 150 $\mu g/m^3$/24 h of particulate matter exposure (104). Exercise should not be done outdoors by those with cardiovascular or respiratory disorders when these first-alert levels of adverse air quality are reported (103). As a general precaution for greatly industrialized or densely populated urban areas, exercise prescriptions for impaired individuals should focus on indoor exercise.

Exposure to Various Interstressors

Of the various environmental stressors discussed in this chapter, only the physiologic responses during exercise to the combined effects of environmental heat and adverse air quality, the combined effects of hypoxia (high altitude) and adverse air quality, and the combined effects of environmental cold and high altitude have been reported. The combined effects of heat stress and air pollutants (specifically CO, O_3, PAN, and PANCO) have been evaluated. Only O_3, however, has been systematically studied at a variety of ambient temperatures. All pollutants (except for one series of experiments in which maximal exercise responses to CO, PAN, and PANCO were evaluated) have been studied at only low exercise intensities (35–40% $\dot{V}O_{2max}$) in the heat. The combined effects of high altitude and CO were reported in cardiovascularly impaired individuals, and the combined effects of O_3on adult hikers at Mt. Washington were studied. Much of the information on cold temperatures and altitude results from the prevalence of these colder temperatures in the high mountains rather than by specific experimental design per se. Generally, the physiologic responses during exercise to cold and altitude have been evaluated from brief exposures to the extremes for these two conditions. To date, few studies have assessed the effects of any of these interstressors on individuals with specific cardiovascular or respiratory disorders.

ENVIRONMENTAL HEAT, HYPOXIA (HIGH ALTITUDE), AND ADVERSE AIR QUALITY

Because the level of many air pollutants is high during periods of air stagnation and is often accompanied by elevated T_a, individuals with an impaired cardiovascular or respiratory system are at even greater risk. Submaximal exercise performance (40% $\dot{V}O_{2max}$) during O_3 exposure (0.50 ppm) was evaluated at different environmental conditions (25°C, 45% rh; 31°C, 85% rh; 35°C, 40% rh; and 40°C, 50% rh). There was a trend toward greater impairment in pulmonary function during combined exposure to O_3 and heat stress (111). Decrements in pulmonary function after exposure to ozone and heat were greatest immediately after exercise. Reductions in vital capacity and maximal voluntary ventilation were significant during the most extreme heat exposure (40°C, 50% rh); exercise $\dot{V}_E$ was highest at this T_a (plus ozone). Because heat and O_3 exposure were not related to additional reductions in any flow variables compared with O_3 alone, other unknown mechanism(s) besides bronchoconstriction probably are related to the decrements in pulmonary function during exposure to the combined stresses.

Although other air pollutants (CO, PAN, and PANCO) have been evaluated during exercise-heat stress, the environmental conditions were limited to 30% rh at 25°C and 35°C (106,108,110,116) during maximal and submaximal (35% $\dot{V}O_{2max}$) exercise intensities. $\dot{V}O_{2max}$ was not altered during exposure to CO, PAN, or PANCO at 35°C. While breathing filtered air, exposure to 35°C lowered $\dot{V}O_{2max}$ (~4%) more than exposure to either single pollutant or the two in combination at 25°C. Although no significant changes in physiologic responses were reported while breathing CO, PAN, or PANCO at 35°C during submaximal exercise, subjective complaints were greater, particularly for PAN and PANCO. Drinkwater et al. speculate that the combination of CO and heat stress is important in the more pronounced respiratory disturbances seen at this elevated temperature (108).

Because these two environmental stressors (heat and pollution) pose additional risks when presented separately to the exercising individual with an impaired cardiovascular or respiratory system, it is not surprising that the risk is potentiated when they are combined. Information available suggests that individuals with limited cardiac or pulmonary reserve capacities are at greater risk during combined exposure to environmental heat plus either CO or O_3. Because evidence is limited, it is premature to conclude that other pollutants do not adversely affect

exercise performance at elevated T_a, particularly in those at risk. Therefore, outdoor physical exercise should not be prescribed for individuals at risk when heat stress levels necessitate caution for healthy individuals or when the current primary federal standards for air pollutants are exceeded (103).

Recently, men with CHD performed exercise stress tests at a simulated 2.1-km altitude while exposed to CO (COHb = 4.2%). The time to onset of angina was reduced by 18%, and there was greater susceptibility to ventricular ectopy than at sea level (126,127). Hikers aged 18 to 64 years climbed Mt. Washington while exposed to low levels of O_3, fine particulate matter, and strong aerosol acidity (128). Those with asthma or wheeze had significantly greater changes in pulmonary dysfunction.

ENVIRONMENTAL COLD AND HIGH ALTITUDE

As one ascends mountainous terrain, both T_a and humidity decrease, with corresponding increases in wind velocity and solar radiation. Although there is considerable variation, T_a decreases ~1°C for every 150 m of ascent (129). The low humidity seen at high altitude promotes increased heat loss through more effective evaporative cooling. Low humidity combined with high pulmonary ventilation can markedly increase heat loss, with serious performance consequences (130). The increased wind velocity decreases the effective temperature at the skin surface. The effects of this wind-chill factor are of particular consequence for preventing peripheral freezing cold injuries due to cooling of exposed surface areas. Increased wind velocity may hamper locomotion, elevate $\dot{V}O_2$, and contribute to fatigue or exhaustion (129). In addition, wind penetration of clothing disturbs the trapped dead air layer and decreases insulation. Although precautions must be taken to prevent sunburn damage, solar radiation provides a necessary source of heat. In contrast to hot environments, it seems important to wear dark colors at altitude, because black clothing absorbs 88% of the solar radiation, khaki 57%, and white only 20% (129).

In addition to selecting dark clothing at altitude, three basic principles for clothing design are suggested to help reduce heat loss (130). They are (*a*) trapping air within clothing and using its insulative properties plus that of the fabric to reduce heat loss, (*b*) using multiple-layered clothing that helps to maximize the use of the entrapped air layer and allows removal or addition of clothing layers as needed, and (*c*) layering heavier and less permeable clothing over more coarsely woven clothing to reduce dampness and heat transfer. The clothing must be kept as dry as possible because wet clothing (from either sweat production or environmental moisture) reduces its insulative properties and results in increased heat loss.

The interactive effects of cold and hypoxic stresses on exercise performance are not well understood, principally because of a lack of experimental information. Ward suggests a number of factors that may decrease exercise performance (129). Cold and hypoxic stresses combined may decrease mental function and could alter exercise performance. Because $\dot{V}O_{2max}$ is reduced at altitude, heat production during exercise is limited at altitude and therefore is associated with a greater risk of cold injury. The increased $\dot{V}_E$ at altitude increases heat and water loss, both of which are an obvious disadvantage. Both cold and hypoxic stress are associated with hemoconcentration and a possible additive increase in blood viscosity, leading to decreased blood flow. Severe peripheral vasoconstriction (cold stress) and high blood viscosity (enhanced by dehydration at altitude) can lead to impaired tissue perfusion and possible tissue necrosis. Because skin blood flow is reduced by hypoxic vasoconstriction at altitude in thermoneutral temperatures, this response may magnify the reduction during cold exposure. Cold and hypoxic stresses may increase lactic acid production for a given exercise intensity, possibly complicating exercise performance. Finally, because the combined stress can produce a life-threatening situation for the normal, healthy individual during exercise, the added risks to those with disorders of the cardiovascular or pulmonary systems are obvious.

HYDRATION IN HOT, COLD, AND HIGH-ALTITUDE ENVIRONMENTS

Hot Environments

Water deficits develop because of fluid nonavailability or a mismatch between thirst and sweat loss. Hypohydration increases T_c responses during exercise in both temperate and hot climates (16,131); fluid

deficits as small as 1% of body weight can elevate T_c and HR during exercise (16,131,132). The T_c rises 0.1 to 0.23°C, and HR increases ~6 beats·min^{-1} per 1% body weight lost (131,133,134). As the water deficit increases, there is a concomitant graded increase in physiologic strain during exercise-heat stress (131,133), and heat tolerance is reduced (131). When exercise is expected to cause fluid losses >2% body weight, target HR prescriptions should be modified. Besides elevating T_c and HR responses, hypohydration negates the thermoregulatory advantages conferred by high aerobic fitness and heat acclimation (135,136). Because thirst does not develop until modest dehydration (1–2%) is present, exercise prescriptions for hot climates should include recommendations to drink early and often and to replace fluids at a rate that approximates sweat loss (137). Intake should not exceed 1.5 L·h^{-1} for extended durations of heavy sweating (14,15).

Cold and High-Altitude Environments

As air temperature decreases, saturation vapor pressure also declines. Thus, cold air has less water content than warmer air, even if the relative humidity is the same. Breathing cold air may increase body fluid loss during exercise because more respiratory water is required to humidify the inspired air as it passes into the lungs. This fact becomes especially important during exercise when $\dot{V}_E$ is increased and at altitude during both rest and exercise (relative to sea level), since hypohydration is a hallmark of successful altitude acclimatization (138). Standard calculations (139) show that respiratory water loss can be ~50% greater during exercise ($\dot{V}O_2 = 3$ L·min^{-1}) when breathing cold (0°C) rather than hot (35°C) air of similar relative humidity (50%). Although short-term respiratory water losses in cold environments can range widely (10–100 mL·h^{-1}, depending on $\dot{V}_E$), these losses can add considerably to total fluid requirements if extrapolated over a prolonged outdoor exposure. In addition, thirst is blunted in cold weather (140), and fluid may be voluntarily restricted to avoid the need to urinate outdoors in freezing temperatures. Because sweating rates can exceed 1 L·h^{-1} during work in cold environments, especially when heavy clothing is worn (140), dehydration can be a problem in cold, high-altitude environments. It is also suspected that peripheral cold injury may be exacerbated by dehydration (141), possibly through an impaired vasoconstrictor response to cold (142). Exercise prescriptions for cold, high-altitude environments must also consider the importance of proper fluid replacement strategies.

Acknowledgment

The authors gratefully acknowledge Terry Hovagimian for her technical assistance in manuscript preparation. The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation.

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